Hypertension: Ophthalmologist's perspective
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Introduction
Among the vascular diseases affecting the eye hypertension is an important concern for the Ophthalmologists. It is one of the leading causes of ocular morbidity. Ocular changes induced by hypertension may be the initial finding in an otherwise asymptomatic patient necessitating a primary care referral. On the other side, a symptomatic patient may be referred to the Ophthalmologist for visual changes caused by hypertension.

Purposes of this article
To arouse interest about the ocular involvement in Systemic diseases in general & Hypertension in particular.
To highlight the importance of meticulous eye examination in all situations.
To stimulate the Non ophthalmologists in realizing the role of fundus examination in the diagnosis and confirmation of their clinical findings.
To urge the Ophthalmologists to examine the eye not treating it an isolated organ but as a part of the whole system...

In this context, we can remember William B. Bean, a renowned medical scholar who described "The Eye as the Gateway to Medical Wisdom."
Peter Mayer Latham, an ancient English clinician used to teach his students - "If you desire to make pathological knowledge the ground work of your credit and usefulness through life, let me advice you not to allow the period of your pupilage to pass by without making a special study of "Diseases of Eye", Here you see almost all disease in miniature; and from the peculiar structure of the Eye, you see them as though through a glass; and you learn many of the little wonderful details in the nature of the morbid processes, which, but for the observation of them in the eye, would not have been known at all. Let every one of you who has a few months to spare give them to the Eye Infirmary".

Basis of ocular manifestations of hypertension

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Hypertension may follow diversification in its presentation or straightforward it may cause some oculopathies. Here, few of its common presentations are being discussed.

Hypertensive retinopathy
- Vascular changes: Persistent hypertension causes - arteriolosclerosis, hyalinization, & vasospasm.

Consequently
- Light reflex change: Broadening of light reflex, copper wiring, silver wiring.
- Arterial attenuation: Focal or generalised. Vascular spasm leads to narrowing which can become permanent by fibrosis.

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Figure 1: Arterial attenuation macular star and cotton wool spot

c. **A V nicking**
Due to compression of hard artery an veins (sharing common adventitia) - **Salus' sign:** deflection of Veins at crossing site. Bonnet's sign: Banking of Vein (dilated) distal to the crossing site. **Gunn's sign:** Tapering of vein on either side of crossing.

• Extravascular changes
  a. **Microaneurysms**
  Most visible by FFA (Fundus Fluorescin Angiogram). Occur at localized areas of capillary wall weakness. Stasis engorgement of the capillary lead to anoxia, poor nutrition contributes to microaneurysm formation.
  b. Retinal haemorrhages
  Streak hges in the nerve fiber layer (predominate) & blot hge in the deeper layer.
  c. Retinal & macular oedema
  Either of transudation of choroidal fluids after breakdown of RPE (Retinal pigment epithelium) or failure of autoregulation of retinal capillaries.
  d. Retinal lipid deposits (hard exudates)
  Either in a scattered pattern or a macular star (Predominant) (due to the radially oriented nerve fibre layer of Henle).
  e. Cotton wool spots
  Due to focal nerve fibre infarcts.
  f. Focal intra-retinal periarteriolar transudates (FIPT)
  Punctate, white opacities, round or oval, alongside major arterioles in deep retinal areas and on disc. They may appear and dislcppear in crops, leave no permanent change.

  g. **Retinal macroaneurysm,** rarely.

• **Changes in malignant hypertension**
  Necrosis and fibrinoid deposition in the vessel wall occurs, (more in choroidal than retinal arteries). Oedema occurs from breakdown of barrier at RPE and failure of auto regulation of retinal capillaries. Papilloedema develops.

Figure 2: Malignant hypertension, reflecting papiloedema, retinal oedema, cotton wool spots, haemorrhages

**Hypertensive choroidopathy**
Choroidal arteries run a relatively short course, hence, systemic pressure is felt stronger than in retinal arteries. Pressure and flow is higher in macula than periphery.
Acute and severe hypertension alters choriocapillaries, resulting in choroidal ischaemia, Retinal oedema, CME (Cystoid macular oedema), Serous RD (Retinal detachment), RPE changes.

It occurs more prominently in young patients with severe acute hypertension, pheochromocytoma, eclampsia, etc.

**Elschnig's spot:** Focal infarcts in the choriocapillaries cause yellow spots in the overlying RPE. They are completely obstructed terminal choroidal arterioles and fibrin and necrotic tissue. They are also known as acute focal RPE lesions and window defects.
Siegrist's streaks: Radially oriented chains of pigmented spots along sclerosed choroidal vessels representing linear foci of RPE disruption, an indicative of malig. hypertension.

Serous RD: Decompensation of the RPE in hypertensive choroidopathy leads to breakdown of the outer blood-retinal barrier, leading to subretinal accumulation of protein-rich exudates -> Serous RD.

Vascular occlusive disorders
A. Retinal artery occlusion
CRAO (Central retinal artery occlusion)
It is usually the result of atheroma, although may be caused by emloli. It results in infarction of the inner two-thirds of the retina, reflex constriction of the whole retinal arterial tree, stasis in retinal capillaries. The central infarct is ischaemic; therefore unlike RVO, hge is minimal. The retina appears white as a result of cloudy swelling caused by intracellular oedema. Fovea is devoid of this change. Normal orange reflex of choroid seen through fovea in contrast to surrounding opaque retina, gives rise to a 'Cherry- red spot' appearance. FFA shows extreme delay in arterial filling and masking of choroidal background fluorescence by retinal oedema. Usually patient has painless total or near total loss of vision unless the pt. retains central vision via a choroidal artery (present in 15-30%eyes) supplying the papillomacular bundle.

BRAO (Branch retinal artery occlusion)
It presents with an acute and severe altitudinal visual field defect. Whitish appearance due to retinal infarction in the distribution of the affected vessel.

Ophthalmic artery occlusion
Vision of no light perception indicates choroidal ischaemia, due to OA (Ophthalmic artery) occlusion in addition to CRAG. Intense retinal whitening is seen. FFA shows marked defect in choroidal & retinal perfusion.

Non- ischaemic CRVO
Mild tortuosity & dilatations of veins. Hges seen throughout the fundus. (not massive). Mild disc swelling. Little or no cotton wool spot. FFA-shows venous stasis, good retinal capillary perfusion. Mild reduction of V / A.

Ischaemic CRVO
Marked reduction of V / A, marked tortuosity & dilatations of veins. Massive hges all over the fundus. Cotton wool spots are common. Disc markedly swollen. FFA-after disappearance of hge-reveals extensive areas of capillary nonperfusion.

BRVO-(Branch retinal vein occlusion)
It may occur near the optic disc and the AV (Artero-venous) crossing site. Blockage of superior temporal vein frequently involves the macula.

Prognostic factors in RVO (Retinal vein occlusion)
Macular oedema -Retinal ischaeemia - Neovascularization - Neovascular glaucoma.

C. Carotid occlusive disease Amaurosis Fugax
Emboli that temporarily obstruct the Ophthalmic or central retinal artery may produce sudden, severe, painless, transient loss of vision. It is called amaurosis fugax. The attack usually lasts for few minutes, then returns to normal as the embolus travels away. Most common source of emboli is fibrin and cholesterol from ulcerated plaques in the wall of the carotid artery.
Ocular ischaemic syndrome
Significant carotid stenosis can result in ocular ischaemic syndrome. Rarely develops in young pts. Males are at greater risk. Bilateral involvement in 20% cases.

Presentations
Vision loss, ocular pain -secondary to ischaemia of the globe or NVG (Neovascular glaucoma), robeosis iridis, ocular hypotony (sometimes).
Retina shows - arterial constrictions, venous dilatations, hges, oedema, cotton wool spots, neovascularization, etc.

Hypertensive neuropathy
A. Non arteritic AION (Anterior ischaemic optic neuropathy)
Infarction of ON(Optic nerve) head results from occlusion of the post. ciliary arteries produces ischaemia+ reduced axoplasmic flow >disc swelling. It is followed by optic atrophy.
B. Neuro- ocular palsy
There may be isolated nerve palsy or may be associated with other neurological deficits. Cranial nerve palsy presents with : Ocular malalignment, diplopia, exposure keratopathy, neurotropic keratopathy, ptosis, etc.

Figure 4: Right sided ptosis and failure of adduction indicating right 3rd nerve palsy

Vascular accidents of brain & brain stem
It may present with various types of neurological field defects according to involvement of various part of visual pathway upto visual cortex (due to vascular accident). Visual problems may be associated with other motor or sensory neurological deficits.

According to neurological deficit actual site of vascular accident can be localized.

Figure 5: CT-scan showing occipitalhaematoma producing homonymous hemianopia

Rupture of Aneurysms
Hypertension may be the cause of formation and rupture of aneurysms. Rupture of carotid aneurysm may present as bilateral pulsatile proptosis due to carotid-cavernous fistula

Fig.5: Corkscrew conjunctival vessels in carotid-cavernous fistula.

Surgical complications due to hypertension
If hypertension is not managed well pre-operatively, surgery may be complicated with excessive haemorrhage (OCR operation), subconjuntival hge., hyphaema in intraocular surgery. Per-operative hypertensive encephalopathy, even cardiac complication is not unusual.

Conclusion
'Act locally, think globally' -like this modern concept of global village, an ophthalmologist, though working in ophthalmic field, should always think to correlate an ocular problem with the whole system and be familiar with the systemic examinations. Side by side, a
non-ophthalmologist while dealing with any systemic problem should give emphasis on ocular examination as in most instances eye is the mirror of the whole system. Again I am to quote from W.B. Bean -

"Nobody has ever really worked out the geography of the Great Republic of Medicine - If he would have, he would definitely realize that the eyes have a head, and the head has a body and the body has a head, and the head has eyes, and these are all part of a greater unity."

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